

was evaluated on initial and delayed images in 4 hours tracer injection. The washout rate of myocardial MIBG (WR) was also obtained from these data using relative decrease in cardiac activity measured at 15 minutes and 4 hours.

Results: Patients were divided into group A (n=36) genotyped Arg389, and group B (n=18) genotyped Arg389Gly or Gly389. No significant differences were found with regard to medication use including β -blockers between group A and group B (18% vs 6%). Initial and delayed H/M in group A were not statistically different from those in group B (initial H/M: 2.04 ± 0.06 vs 2.13 ± 0.06 , delayed H/M: 1.82 ± 0.06 vs 2.01 ± 0.08). WR was significantly higher in group A than in group B ($48.4 \pm 2.2\%$ vs $38.3 \pm 3.6\%$, $P=0.02$). Left ventricular function estimated by EF was not different between 2 groups ($31.6 \pm 1.9\%$ vs $31.1 \pm 2.4\%$).

Conclusion: Our findings suggest that Arg389Gly polymorphism in β 1AR gene is associated with attenuated CS nervous system activity, which can be the predictor of favorable β -blocker response, in patients with DCM. The Arg389 DCM patients may be good candidates for β -blocker therapy.

1119-64

Functional Fate After Revascularization of Myocardium With Intact Perfusion but Without Contractile Reserve

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It is currently unclear whether myocardium with intact perfusion but without contractile reserve will improve in function post-revascularization. Therefore, we evaluated patients with chronic ischemic left ventricular (LV) dysfunction before revascularization with myocardial perfusion imaging and dobutamine stress echocardiography. Thus, 108 patients with ischemic cardiomyopathy (LVEF $34 \pm 10\%$), scheduled for CABG, underwent both resting tetrofosmin SPECT (to evaluate resting perfusion) and dobutamine echocardiography (to evaluate contractile reserve). Resting LV function was evaluated before revascularization, and late (9-12 months) post-revascularization by 2D echocardiography (18-segment model). Before revascularization, 1336 segments were dysfunctional. Improvement in function was observed in 357 (27%) dysfunctional segments, whereas 979 dysfunctional segments did not improve. The majority of the segments with improvement post-revascularization exhibited both preserved perfusion and contractile reserve (66%). The majority of the segments without improvement of function post-revascularization did not have preserved perfusion or contractile reserve (58%). Of interest, 22% of the segments with improvement and 25% of the segments without improvement showed preserved perfusion but did not have contractile reserve. Thus, dysfunctional myocardium with preserved contractile reserve and intact perfusion has a high likelihood of functional recovery post-revascularization; segments without contractile reserve/perfusion have a low likelihood of recovery. Segments with preserved perfusion but without contractile reserve have an intermediate likelihood of recovery.

1119-65

Comparison of Fluorodeoxyglucose Positron Emission Tomography and Nonfluoroscopic Electroanatomical Mapping in Myocardial Viability Assessment

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Background. Nonfluoroscopic electroanatomical mapping is a novel technique that enables to acquire an on-line information on electrical and mechanical signal of myocardial tissue in the catheterization laboratory. The aim of the present study was to compare the diagnostic value of NOGA electroanatomical mapping with fluorodeoxyglucose positron emission tomography (FDG-PET) and resting and late resting 201-Thallium myocardial perfusion scintigraphy in the myocardial viability assessment.

Methods. Twenty patients with ischemic coronary artery disease and stable angina pectoris underwent electroanatomic mapping, FDG-PET and 201-Thallium resting-late resting scintigraphy. After exclusion of 5 patients with perfusion metabolism mismatch, data on 15 patients (80% male, 61 ± 11 y) were analysed. The quantitative FDG-PET and Thallium uptake data were calculated by polar map analysis by division into 12 comparable myocardial segments, as represented in the electroanatomic mapping image. **Results.** Significant logarithmic correlation was found between NOGA unipolar voltage and FDG uptake values ($r=0.685$, $p<0.001$) as well as late resting Thallium-201 uptake ($r=0.797$, $p<0.001$). Nonviable myocardial segments (late resting Thallium-201 uptake $<50\%$) exhibited 4.7 ± 1.9 mV endocardial potentials and $39.9 \pm 10.3\%$ FDG uptake, while ischemic but still viable (late resting Thallium-201 uptake between 51-75%) and normal myocardium (late resting Thallium-201 uptake $>75\%$) displayed 8.6 ± 3.7 mV and 13.5 ± 4.8 mV endocardial voltage potentials and 65 ± 13.5 and $76.4 \pm 9.9\%$ FDG uptake. A good concordance between unipolar voltage and FDG-PET values was observed in the nonviable myocardial areas. However, in myocardial territories of normal and mild reduced viability, unipolar voltage values exhibited greater scattering, leading to an underestimation of the myocardial viability in these segments. **Conclusion.** Despite a global good correlation between endocardial unipolar voltage values and FDG uptake, it seems, that NOGA electroanatomical mapping overestimates the severity of resting myocardial ischemia in comparison with FDG PET.

1119-66

Left Ventricular Functional Impairment in Chronic Heart Failure Patients Is Independent of Extent of Perfusion Defects and Viable Myocardium

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Purpose: We tested the hypothesis that the left ventricular ejection fraction (LVEF) in chronic heart failure (CHF) patients declines as a function of the perfusion defect size. If true, then the fraction of hypoperfused but viable myocardium could predict a post-revas-

cularization increase in LVEF.

Methods: In 104 CHF patients, an LVEF of $24 \pm 7.3\%$ (range 10 to 40%) and referred for cardiac transplant evaluation, the sizes of mismatch (MM, viable) and match (M, scar) myocardium on rest perfusion (13N-ammonia) and 18F-deoxyglucose PET images were determined by quantitative polar map analysis (Munich Heart Analysis) in % LV correlated with the LVEF determined clinically by angiography, radionuclide imaging or 2D-echocardiography.

Results: Perfusion defect sizes (sum of MM and M), ranged from 10 to 40% (avg. 24 ± 7.3), and did not correlate with LVEF ($R^2=0.01$). There was also no correlation for LVEFs by angiography only, excluding a method related variability in LVEFs. Similarly, no correlation was found for LVEFs with primarily MM (*40% mismatch and -20% of match, n:13, 20.7 ± 6.7 , $R^2=0.08$) or primarily M (*40% match and -20% of mismatch, n:10, LVEF 23.2 ± 8.1 , $R^2=0.07$) defects. Neither stress induced ischemia (n= 25, LVEF 24 ± 7.0 , $R^2=0.01$) nor mitral regurgitation (n=4, LVEF 26 ± 9.4 , $R^2=0.01$), diabetes (n= 28, LVEF 26 ± 6.1 , $R^2=0.02$) and arterial hypertension (n=32, LVEF 23 ± 7.2 , $R^2=0.01$) influenced the lack of correlation between LVEF and perfusion defect size.

Conclusions: In this highly selected CHF population, the impairment of LVEF was independent of the extent of perfusion defects or hypoperfused but viable myocardium, stress induced ischemia or diabetes or arterial hypertension, and most likely resulted from LV remodeling. This suggests that the extent of hypoperfused but viable mismatch myocardium is limited in predicting the degree of post-revascularization a LVEF improvement.

POSTER SESSION

1139 Experimental Studies With Myocardial Contrast Echocardiography

Monday, March 18, 2002, 3:00 p.m.-5:00 p.m.

Georgia World Congress Center, Hall G

Presentation Hour: 4:00 p.m.-5:00 p.m.

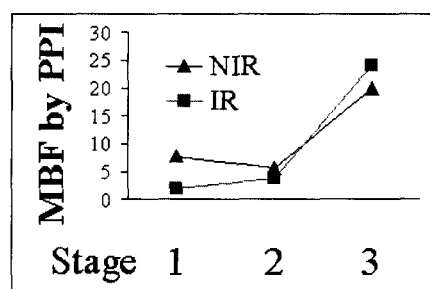
1139-53

Insulin Improves Myocardial Microcirculation Reperfusion After Acute Ischemia With Hyperglycemia

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We have demonstrated hyperglycemia (HG) worsens myocardial microcirculatory reperfusion (RP) after acute ischemia with power pulse inversion imaging (PPI) and Neutron Activated Microspheres (MIC). To test the hypothesis that insulin can improve myocardial RP, we studied myocardial contrast enhancement during a continuous intravenous infusion of PESDA microbubbles in 6 dogs for 3 sequential stages: 1) during coronary ligation, 2) following reperfusion in the presence of HG (plasma concentration of 321 ± 89 mg/dl), 3) following insulin intravenous injection with normal plasma concentration. Left ventricle was imaged on short-axis at mid-papillary muscle level. Ischemic and non-ischemic (IR, NIR) regional myocardial blood flow (MBF) was estimated by the product of the rate of replenishment and the plateau of myocardial acoustic density at the regions. MIC was used for validation. **Results:** 1) PPI-derived MBF correlated well with MIC-MBF ($r=0.91$, $p<0.0001$). 2) During stage 1, MBF was 1.98 ± 1.61 in IR, 7.81 ± 5.46 in NIR. 3) During stage 2, MBF only mildly increased in IR (3.79 ± 3.33 , $p=0.11$), but decreased in NIR (5.51 ± 6.48 , $p=0.01$). 4) During stage 3, MBF significantly increased both in IR 24.24 ± 11.96 , $p=0.006$ and in NIR (20.0 ± 7.64 , $p=0.0002$, Fig).

Conclusion: Insulin improves myocardial blood flow following reperfusion when hyperglycemia is present. It indicates the importance of intensive insulin therapy for diabetic patients during acute ischemia and reperfusion.



1139-54

Color-Coded Curved Anatomical M-Mode Analysis of Contrast Echocardiography: A Novel Technique for the Diagnosis of Coronary Artery Stenosis

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Background: Replenishment curve of intensity after bubble destruction in real-time contrast echocardiography should be useful for diagnosing coronary stenosis. However, it is hard and time-consuming to calculate the replenishment curve in every region of the ventricular wall. Newly developed curved anatomical M-mode (Camm) provides the temporal change of intensity along an arbitrary setting line.

Purpose: The purpose is to elucidate efficacy of color-coded Camm for diagnosing an area at risk by adenosine triphosphate (ATP) administration.